EXERCISE AND HYPERTENSION

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A number of risk factors seem to play a role in the development of cardiovascular disease, including hypertension, smoking, serum cholesterol levels, obesity and a sedentary life style. It is encouraging, therefore, that reductions of these risk factors have been shown in several recent studies to have a beneficial effect by reducing the morbidity and mortality associated with cardiovascular disease.

The nationwide public health effort in treatment of hypertension, changes in dietary and smoking habits and the enormous growth in sports participation appear to be related, at least temporarily, to this decline in mortality. While hypertension predominated as a major health focus during the 1970s, physical activity and sports have recently preoccupied much of the lay and medical press. The 1981 National Conference on Fitness and Aging[1] concluded that regular exercise might beneficially alter the observed reduction in functional capacity of the cardiovascular, respiratory and musculoskeletal systems associated with aging. In this regard it is of interest that one of the earliest medical writings devoted to exercise, the *Libro del Exercicio Corporal,*[2] acknowledged that exercise in moderation is important to maintain good health. Published in Seville in 1553, Cristobal Mendez wrote: "The fifth age is old age, which is past forty or more years, and in this one the proper exercise is to ride a mule, or to walk for a while on foot. If one had the habit of doing some of the past exercises...it is good to keep doing it in moderation....The proper exercise for this and the sixth age, which is decrepitude from seventy years on, is gentle movement and use of temperance in everything he has been used to."

EPIDEMIOLOGIC STUDIES

Several lines of evidence do in fact provide a theoretical basis for exercise conditioning as a way to lower blood pressure and to reduce the risk of cardiovascular disease. Many epidemiologic studies have suggested the

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salutary effect of regular physical activity. An early study by Morris et al.[3] of London transport system and post office workers assumed that a level of activity could be correlated with a specific job. In this study, sedentary bus drivers and telephonists had a greater rate of cardiovascular disease than their more active counterparts, conductors and postmen. Paffenbarger and Hale[4] likewise noted a lower risk of fatal heart attack among longshoremen involved in tasks associated with high levels of physical activity. Similar results were obtained in a separate study of 16,936 Harvard alumni[5] as well as in the prospective Framingham Study.[6] It is of note that in the Harvard alumni study[5] a lower risk of heart attack was observed in those individuals who maintained a high level of physical activity such that they utilized greater than 2,000 kilocalories per week. This reduction in heart attack risk was notably independent of their level of athletic involvement as undergraduate students.

With respect to blood pressures, several epidemiologic studies have observed that active individuals have lower blood pressures. Montoye et al.[7] assessed the habitual physical activity of 1,700 men 16 years old and older. Both systolic and diastolic blood pressures were significantly lower in the more active men. Taylor[8] likewise noted a difference in blood pressure between active railroad switchmen and more sedentary clerks. This study observed that blood pressures were similar at the beginning of employment, which suggests that preselection could not explain the observed differences in blood pressures later in life.

Blood pressures have been noted, in some studies, to be lower following exercise training in normotensive individuals,[9,10] but this finding has not been reported by all investigators. It should also be noted that body fatness has generally been correlated with resting blood pressure levels. Montoye,[7] among others, found that body fatness was higher in sedentary men. As a potentially confounding variable, changes in weight must be considered when studying the effects of physical training on blood pressure.

Unfortunately, many of these epidemiologic studies suffer from methodological problems that make it difficult to conclude that regular exercise is associated with a reduction in blood pressure. Furthermore, we cannot conclude that physical inactivity by itself is a risk factor for the development of cardiovascular disease.

Animal Studies

As in other areas of research, animal models have been studied to attempt to clarify the role of exercise in reducing blood pressure. Tipton and his coworkers[11] exercise trained normotensive, borderline hypertensive and spontaneously hypertensive rats. Following 10 to 12 weeks of training, the normotensive rats had lower blood pressures than nontrained control rats. In the borderline hypertensive rats and spontaneously hypertensive rats, training likewise lowered blood pressures and blunted the rise in pressure as the spontaneously hypertensive rats matured. A somewhat disturbing finding was the observed increase in heart weight in trained spontaneously hypertensive rats. Whether or not such changes occurring in hypertensive human beings with underlying myocardial hypertrophy and dilatation would prove detrimental remains speculative.

A similar attenuation of blood pressures was observed by Shepherd et al.[12] within 3-4 weeks of training Dahl salt-sensitive rats. Running at 20 m/min, 60 min/day, 5 days/wk prevented a rise in blood pressures over 12 weeks in these salt-free Dahl-S rats. After 24 weeks, blood pressures rose despite continued training but remained below those of nontrained Dahl-S rats. Delaying training until after the development of hypertension prevented any exercise-induced reduction of blood pressures, a finding which raises a question as to the anticipated benefit of exercise training in human subjects with fixed hypertension.

While several other groups have observed a reduction of blood pressure in hypertensive trained rats, the finding is by no means universal. It is likely that such factors as type, intensity and length of training as well as the model for hypertension play a role in the lack of agreement between the various studies. There is, of course, always the problem of using data from an animal model when studying a disease of humans where the pathophysiology of the model may be quite different.

Human Studies

Observations in humans have, as noted earlier, indicated that active individuals have lower blood pressures than their sedentary counterparts. Several prospective studies have tended to support this impression.

Studying the effect of six months of exercise training in 23 hypertensive men involved in a two-day-per-week walk-jog program, Boyer and Kasch[13] demonstrated a mean drop in systolic blood pressures of 13.5 mmHg and in diastolic blood pressures of 11.8 mmHg. Normotensive exercise controls experienced a 6 mmHg fall in diastolic pressure with no change in systolic blood pressure. Choquette and Ferguson[14] demonstrated a similar effect in 37 borderline hypertensive subjects. Following six months of home calisthenics, there was a mean reduction at rest of 15 mmHg in systolic and

8 mmHg in diastolic blood pressures. During exercise, blood pressure rose appropriately from values at rest but remained lower than preconditioning values. The group from the Institute of Sports Medicine in Prague[15] conducted a series of hemodynamic studies in 10 male hypertensive subjects with a mean systolic blood pressure of 182 ± 4.9 mmHg and diastolic blood pressure of 99 ± 1.6 mmHg. Intraarterial blood pressure measurements before training demonstrated a rise in both systolic and diastolic blood pressures during exercise with a recovery to baseline values in 10 minutes postexercise. Following a brief one month bicycle training program, there was no apparent effect on resting blood pressures. However, the rise in blood pressures during exercise was significantly blunted. In addition, there was a reduction in tension time index and rate pressure product, both of which are indices of myocardial work. A similar reduction in blood pressures and submaximal rate pressure product was demonstrated by our group while studying the response of exercise conditioning in renal dialysis patients.[16] Beginning at a mean blood pressure level of $170 \pm 14/99 \pm 2$, dialysis patients participating in a supervised three day/wk exercise program experienced, in five weeks, a significant reduction in blood pressures, with a mean decrease of 22 mmHg in systolic and 11 mmHg in diastolic blood pressures (Figure 1). A nonexercised control group of dialysis patients had no significant change in blood pressures. Exercise training also resulted in a lower rate pressure product at a similar level of submaximal oxygen consumption (Figure 2). Since myocardial oxygen demand is linearly correlated with rate pressure product,[17] at any given level of submaximal effort myocardial oxygen demand was less following 10 weeks of exercise conditioning in hemodialysis patients. It should be noted that the effects of exercise appear to depend on continued participation in the exercise program. As demonstrated in Figure 3, when our patients discontinued the exercise regimen, a prompt elevation in blood pressures was noted.

Bonanno and Lies[18] reported a significant reduction of 13 mmHg in systolic and 14 mmHg in diastolic pressures following a conditioning program for hypertensive subjects. The magnitude of change, while impressive, must be viewed in the context of a simultaneous reduction in diastolic blood pressures of 11 mmHg in a hypertensive nonexercised control group. Kukkonen et al.[19] likewise found that their hypertensive control group had a modest but statistically significant reduction of 7 mmHg in diastolic blood pressures. In contrast, the exercise-trained subjects exhibited not only a significant reduction in diastolic blood pressure of 11 mmHg but also in systolic blood pressure of 9 mmHg.

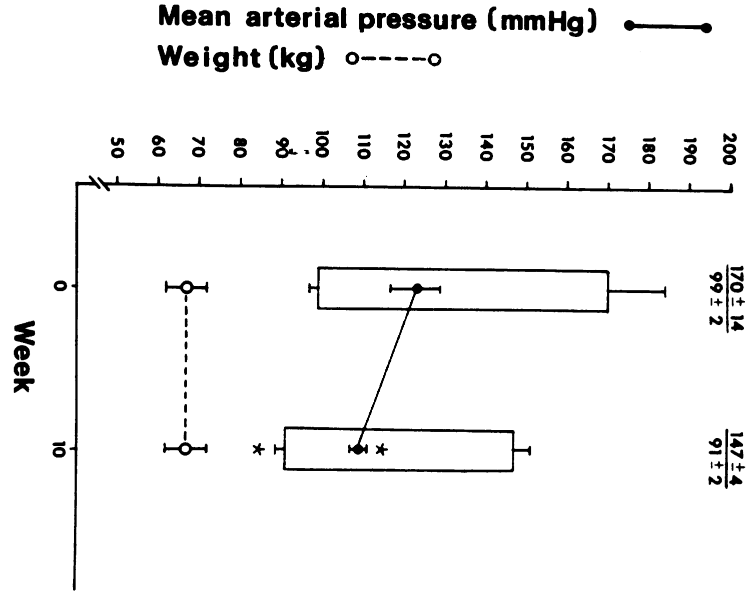

Fig. 1. The blood pressure response to a 10-week exercise conditioning program in five hemodialysis patients. Measurements of systolic, diastolic and mean arterial pressure (closed circles) were obtained at rest in the sitting position before an exercise fitness evaluation at weeks zero and 10. Weight measurements (open circles) represent the mean±SEM of three predialysis values for each patient during the week of observation. P<0.05. Reproduced by permission from Zabetakis, P.M.: Profiling the hypertensive patient in sports. *Clin. Sports Med.* *3*:137-52, 1982.

These findings underscore the need for multiple blood pressure measurements before classifying any patient as having hypertension. Significant reductions in blood pressure without therapy were observed in the Australian Therapeutic Trial on the pharmacologic treatment of mild hypertension.[20] During the trial, untreated hypertensives experienced a 6.6 mmHg fall in diastolic blood pressures from 100.4 to 93.9 mmHg. This was certainly less than the 12.2 mmHg fall in diastolic blood pressure observed in the pharmacologically treated group, yet it is interesting that the magnitude of blood pressure reduction in the untreated group is comparable to that reported above for nonexercised control subjects. In addition, the exercise-induced reduction in diastolic blood pressure (11-14 mmHg) was quite similar to that seen in two recent pharmacologic studies: the Australian Trial (mean 12.2 mmHg) and the mild hypertensives in the Hypertension Detection and Follow-up Program (mean 12.9 mmHg).[21] In the Hypertension Detection and Follow-Up Program study, the pharmacologically induced fall in blood pressure was

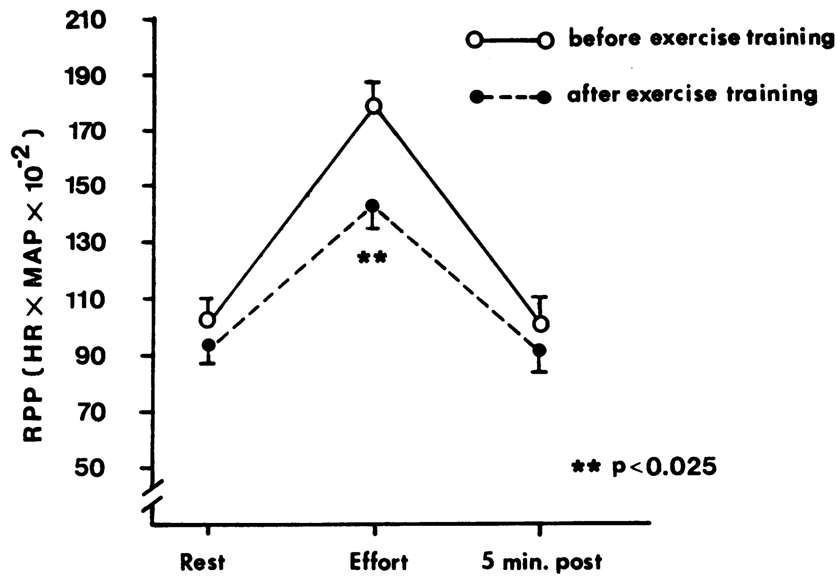

Fig. 2. Rate pressure product (RPP) is shown at rest, at a similar level of oxygen consumption (18.0 ± 1.0 ml/kg/minute) during a treadmill test (effort), and five minutes after effort in five hemodialysis patients. $P < 0.025$. Reproduced by permission from Zabetakis, P.M.: Profiling the hypertensive patient in sports. *Clin. Sports Med.* *3*:137-52, 1982.

associated with a 20.3% reduction in mortality from all causes and 19% fewer cardiovascular deaths when compared to a randomized control group who had only an 8.6 mmHg drop in blood pressure.

While similar data are not available on the impact of an exercise-induced reduction in blood pressure, it is interesting to speculate that a reduction of blood pressure through either exercise or drugs might have a favorable influence on the development of cardiovascular disease and premature death. Paffenbarger et al.[22] recently reported on the influence of physical activity on mortality and longevity among 16,936 college alumni. It is apparent from these data that while hypertension was associated with an increased relative risk of death, death rates declined steadily as the level of physical activity increased. Activity levels in excess of 2,000 kcal per week were associated with a relative risk of dying one half that among individuals with less than 500 kcal of physical activity per week. In as much as exercise reduced the risk of death among hypertensive alumni who remained hypertensive, the authors concluded that it was unlikely that the reduction in risk could be contributed to antihypertensive therapy. These findings are certainly provocative.

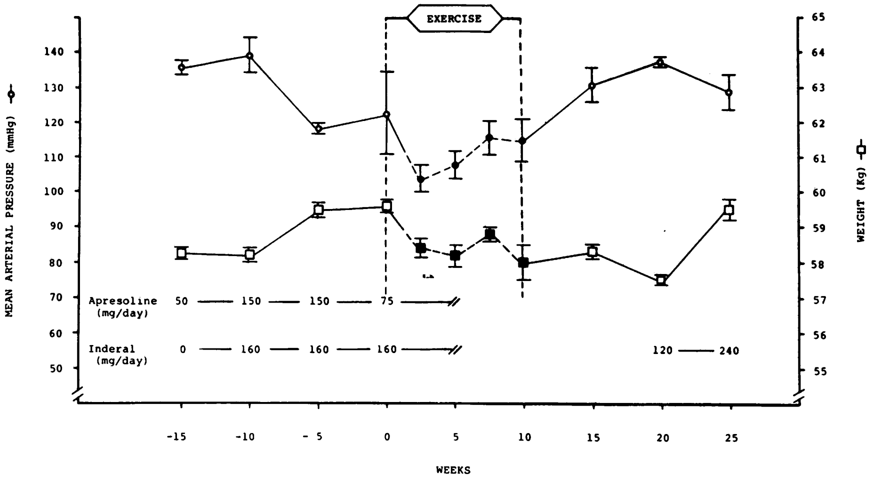

Fig. 3. The blood pressure response to a 10-week exercise conditioning program in a hypertensive hemodialysis patient. Mean arterial pressures and weights represent the mean±SEM of three predialysis measurements during the week of observation. Closed circles and closed squares represent measurements during the exercise-conditioning program. There was no correlation between the changes in blood pressure and weight, Reproduced by permission from Zabetakis, P.M.: Profiling the hypertensive patient in sports. *Clin. Sports Med.* *3*:137-52, 1982.

Summary And Conclusion

While there remains some controversy over the effects of exercise training in hypertensive individuals, epidemiologic studies, animal data and numerous studies in patients with mild hypertension seem to indicate that exercise conditioning can result in a modest fall in blood pressure and a reduction in myocardial oxygen demand. As to the mechanism responsible for these pressure changes, there are no unifying theories. Exercise conditioning is known to result in an increase in blood volume, a reduction in resting heart rate, a slightly lower resting cardiac output and a reduction in myocardial oxygen demands. Considering the measurable hemodynamic changes that occur with physical conditioning, it would appear that an alteration in overall autonomic balance may be responsible for the reduction in blood pressure. A reduction in sympathetic tone; a decrease in baroreceptor sensitivity, an increase in β_2 and decrease in α receptor sensitivity, a change in myotonic tone, a decrease in cardiac output, and alterations in non-neurogenically-mediated vascular compliance may all exert an effect on reducing resting blood pressure in exercise-conditioned individuals.[16]

The intensity and duration of exercise may also be important to the anticipated effect of the conditioning program. Exercise at a sufficient intensity to exceed the lactate threshold has been shown to be associated with a marked increase in the pressor hormones, renin and catecholamines.[23,24] While the physiologic impact of this increase in pressor hormones is as yet not clearly understood, exercise of low intensity and long duration can provide for a reduction in blood pressure and improvement in cardiovascular conditioning without evoking a pressor hormone response.

Exercise-induced reduction in blood pressure of the magnitude reported could certainly impact favorably on an individual's risk factor profile. For patients with mild to moderate hypertension, a supervised exercise program may be considered as a safe and potentially beneficial adjunct to current antihypertensive therapeutic regimens. Under close medical follow-up, exercise could also be employed as an initial nonpharmacologic approach to individuals with mild hypertension. However, the lack of sufficient data from well controlled studies makes it difficult to recommend enthusiastically, on any large scale, the use of exercise conditioning as an alternative to the pharmacologic treatment of hypertension. It is hoped that additional data will be forthcoming that will define the proper role of exercise conditioning in the treatment of hypertensive individuals.

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